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Abstract: Samples were collected in 35 states as part of a national monitoring system to detect multiple diseases in feral swine (*Sus scrofa*). During March 2009 through December 2010, we collected serum samples from 4,479 feral swine from 13 states, and 159 animals tested were seropositive for brucellosis. No difference in likelihood of infection was found between males and females, but adults were more likely than sub-adults or juveniles to be exposed to brucellosis. Feral swine sampled during winter months also were more likely to be seropositive than animals sampled during other seasons. Apparent prevalence varied among states, and seropositive animals often were clustered in specific counties within a state. We recommend improved diagnostics and stricter regulations on movement of feral swine both intra- and inter-state to minimize further spread of the disease and to decrease the risk of re-introduction of brucellosis into livestock.

Key words: *Brucella suis*, feral swine, human–wildlife conflicts, prevalence, *Sus scrofa*, swine brucellosis

FERAL SWINE (*SUS SCROFA*) are considered an invasive species in the United States. For the purposes of this paper, feral swine are defined as free-roaming pigs whose genetic lineage includes escaped domestic swine (*Sus scrofa domestica*), Eurasian wild boar (*Sus scrofa scrofa*), and any hybrids of these 2 subspecies. They are found in at least 38 states (Wyckoff et al. 2009), and a recent estimate suggested a nationwide population of 5 million feral swine (Pimentel 2007). Feral swine are able to tolerate and exploit many environmental conditions, grow and reproduce rapidly; they are aggressive competitors for local resources and lack natural predators other than humans throughout most of their geographic distribution. Consequently, they are responsible for causing extensive ecological damage wherever they are found (Seward et al. 2004).

Feral swine carry a number of endemic diseases that can pose a risk to humans, as well as to cattle and swine operations. One such disease is swine brucellosis, caused by the bacterium *Brucella suis*. There are several recognized species of *Brucella*, and each is associated with a specific animal host. While *B. suis* infects swine primarily, it also can cause disease in cattle (Cook and Noble 1984), horses (Deyoe 1986), dogs (Kerby et al. 1943), and humans (Young 1995, Cvetnic et al. 2005). Similarly, swine also may become infected with *B. abortus* or *B. melitensis*. The primary route of transmission for *B. suis* in feral swine is thought to be venereal, but vertical transmission also has been documented via infected milk or oral exposure to infected tissues, such as aborted fetuses and placental tissues (Deyoe 1986).

The commercial swine industry in the United

States maintains brucellosis-free status in all states, but the presence of brucellosis-infected feral swine populations, and the potential for feral swine to transmit disease to domestic swine (i.e., captive domesticated pigs bred for meat and contained in small or large fenced areas or buildings) could jeopardize the commercial swine industry. Improved understanding of the prevalence and geographic distribution of brucellosis in feral swine is important for informing and guiding relevant management decisions that will help ensure the security of U.S. swine and cattle industries. In addition, feral swine are known to carry other zoonotic *Brucella* species. Brucellosis in humans manifests itself as recurrent fever, chills, headaches, and general weakness, and can afflict those infected for extended periods of time (van der Leek et al. 1993). Hunters, wildlife biologists, and anyone involved in butchering or dressing infected feral swine are at risk (Centers for Disease Control and U.S. Department of Agriculture 2010).

The U.S. Department of Agriculture, Animal and Plant Health Inspection Service, Wildlife Services' (WS) National Wildlife Disease Program implemented a broad-scale surveillance program to provide pertinent information on numerous diseases in feral swine populations. As part of this larger comprehensive surveillance program, we developed a project to assess apparent seroprevalence of brucellosis in feral swine populations, determine if disease status was related to age or sex, examine any seasonality patterns associated with disease exposure, and identify any potential spatial disease clusters with higher than expected levels of seropositivity.

Methods

Sample collection

From March 1, 2009, to December 31, 2010, WS wildlife disease biologists collected samples from feral swine in 35 states. Samples were collected opportunistically from feral swine removed for wildlife damage management purposes or specifically for disease surveillance purposes, and occasionally from hunter-killed animals. Feral swine populations in close proximity to landfills, airports, and other areas that were considered to be high-risk U. S. entry points of foreign animal diseases, such as classical swine fever, were given priority for sampling. Blood samples were collected primarily by cardiac puncture and placed in serum-separating Vacutainer® tubes. Once the blood clotted, it was centrifuged, and the serum was transferred into 2 ml Cryovials® and labeled with a unique barcode number. The serum was shipped to the laboratory on the same day or stored at 4°C and shipped usually within 3 days of collection. Samples that could not be shipped within 3 days were frozen at -20°C and shipped no later than 2 weeks after collection. Samples were shipped overnight with ice packs or dry ice.

Testing procedures

From March 1, 2009, to September 30, 2009, 1,382 samples were submitted to the Kansas State Federal Brucellosis Laboratory (KS-FBL) or the U.S. Department of Agriculture, Animal and Plant Health Inspection Service, Veterinary Services Laboratory in Kentucky. The Kansas Laboratory screened the samples with the rapid automated presumptive (RAP) test (Mikolon et al. 1998). If negative, testing was considered complete; if positive, samples were tested with the particle concentration florescent immunoassay (PCFIA; Davis et al. 1980). The Kentucky laboratory performed 3 tests in series, starting with the buffered acidified plate antigen (BAPA) test (Alton et al. 1988). If this test was negative, testing was considered complete; if positive, samples were tested using the Rose Bengal card test (RBCT; van der Leek et al., 1993). If the RBCT was negative, testing was considered complete, but if positive, samples were tested again using the fluorescence polarization assay (FPA; Nielsen et al. 1996). Samples were considered positive only if they tested positive on all tests performed at either the Kansas or Kentucky laboratory. None of the tests performed is specific enough to distinguish between *Brucella abortus* or *B. suis*; however, they are the best available assays for determining brucellosis exposure and are the accepted method for diagnostic laboratories.

Sample testing procedures were changed slightly beginning October 1, 2009, to make the process more efficient and to standardize diagnostic testing; the RBCT was used at the National Wildlife Disease Program laboratory in Fort Collins, Colorado, to screen 3,097 samples for brucellosis antibodies. Any sample

that tested positive was forwarded to the KS-FBL where the FPA test was performed.

Analysis

Mean seroprevalence and associated 95% confidence intervals were calculated using a binomial distribution for prevalence of *Brucella* spp. in feral swine by state. Potential disease associated risk factors were analyzed using a mixed model (Proc Glimmix) in SAS version 9.1. Data were run using a logistic link function and binary error using antibody presence (positive versus negative) as the outcome variable. Degrees of freedom were calculated using a Kenward-Roger adjustment to account for sample size differences and control for Type I error. All logistic regression factors were categorical and included age (adult ≥1 year; sub-adult = 2 months to 1 year; and juvenile ≤ 2 months; Matschke 1967) and sex of sample animals, as well as season of sample collection (spring = March 20 to June 20; summer = June 21 to September 22; fall = September 23 to December 21; and winter = December 22 to March 19). State location was set as a random variable.

Spatial association of *Brucella* spp. data also was analyzed in SatScan (version 9.0.1), using a Bernoulli model to determine if seropositive feral swine clustered in specific areas, during specific time periods (grouped into 1-month intervals). SatScan generates a spatial scan statistic using a moving circular window, with a base that corresponds to geographic area and height corresponding to time (Kulldorff 1997). As the window moves in space and time, the base varies from 0 to a set maximum radius of 50% of the population, which allows the detection of both small and large clusters. *P*-values are generated by repeating 999 replications of the data set generated under the null hypothesis using Monte Carlo simulation (Kulldorff 1997), with the null hypothesis stating that the number of cases within the window is similar to the number of cases outside the window.

Results

From March 1, 2009, through December 31, 2010, 4,479 feral swine were sampled for brucellosis in 35 states (Figure 1). Most of the samples were collected from adults (2,787), followed by sub-adults (1,180) and juveniles (512); 2,177 samples were collected from males and 2,302 samples were collected from females.

Positive samples were identified in 34 counties of 13 states (Table 1) and apparent prevalence in these states ranged from 0.7 to 14.4% (Table 2). The apparent prevalence of brucellosis was greatest in Alabama, Hawaii, and South Carolina (Table 2). There were 159 feral swine that were seropositive for brucellosis during the study, many of which were collected in relatively few counties (Table 1). All of the seropositive samples were collected from feral swine in the southern and southeastern portions of the United States and Hawaii (Figure 1).

Age category was associated with brucellosis ($P < 0.0001$) and odds ratios revealed that adult animals were 2.8 (Confidence Interval [CI] = 1.7 to 407) times more likely to be seropositive than were sub-adults, and 7.0 (CI = 2.1 to 22.8) times more likely to be seropositive than juveniles. Season of capture also was a significant parameter ($P = 0.01$). Feral swine sampled in winter were more likely to be seropositive than those sampled in spring (Odds Ratio [OR] = 1.50, CI = 0.9 to 2.3), summer (OR = 2.21, CI =

Table 1. Counties where ≥1 feral swine samples were identified as antibody positive for *Brucella* spp.

State	Counties
Alabama	Clarke
Arkansas	Arkansas, Baxter, Desha, Hempstead
Florida	Marion, Pasco, Palm Beach, Orange, Highlands, Polk
Georgia	Glynn, Chatham, Oglethorpe
Hawaii	Honolulu
Kansas	Bourbon
Louisiana	Evangeline
Mississippi	Bolivar, Yazoo
Missouri	Reynolds
North Carolina	Bladen, Johnston
Oklahoma	Choctaw, Jefferson, McCurtain
South Carolina	Calhoun, Georgetown, Richland, Marlboro
Texas	Houston, Freestone, Leon, Liberty, Smith

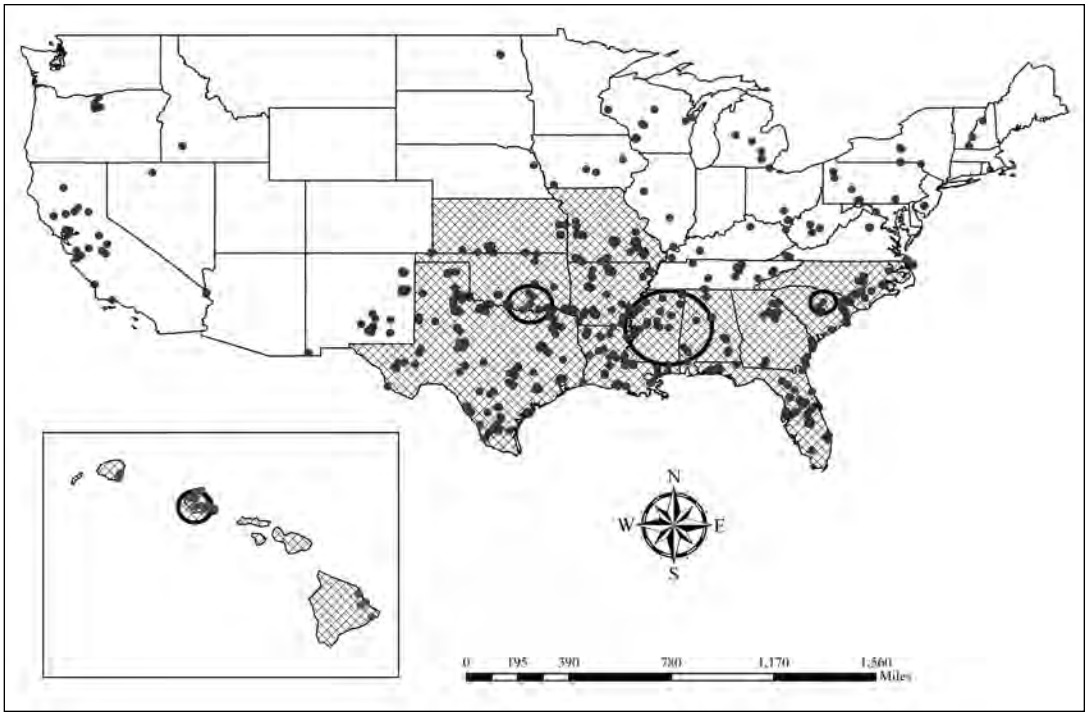

Figure 1. The points represent feral swine collection sites from March 1, 2009, through December 31, 2010, in the continental United States and Hawaii. States containing ≥1 *Brucella* spp. seropositive individual are identified by shading; circles represent locations of swine brucellosis spatial clusters.

1.3 to 3.7), or fall (OR = 1.73, CI = 1.0 to 2.7). In addition, feral swine sampled in spring (OR = 1.47, CI = 0 .8 to 2.4) and fall (OR = 1.278, CI = 0.6 to 2.5) had higher prevalence values than those sampled in summer, although confidence intervals overlapped 0. Sex was not a significant predictor of brucellosis exposure, with males having only slightly higher exposure levels than females (OR = 1.19, CI = 0.85 to 1.6).

Spatial-temporal analyses identified 4 brucellosis clusters within our data set (Figure 1). One cluster with higher than expected case numbers was located in Hawaii on the island of Oahu (P = 0.001), with positive cases occurring throughout the study period. Another large disease cluster included the region of Alabama, Mississippi, Louisiana, and Arkansas. Positive samples in this cluster were identified from February 2010 through March 2010 (P = 0.001). Remaining disease clusters were identified in South Carolina (P = 0.001) with seropositive animals identified throughout the study period, as well as a cluster around a portion of the Texas-Oklahoma border (P = 0.001) that identified a higher than expected number of positives from March to October 2010.

Discussion

Feral swine populations, distributions, and densities are difficult to estimate in the United States (Pimmentel 2007). Little is known about diseases that can be maintained or transmitted by feral swine, such as brucellosis (either *B. suis* or *B. abortus*), but the documented presence of brucellosis in feral swine reported here poses a risk to cattle and commercial swine production. It must be emphasized that these industries in the U.S. are currently free of brucellosis; however, the potential for disease transmission to commercial animals and at-risk humans will remain as long as endemic diseases, such as brucellosis, exist in feral swine.

We screened samples for *Brucella* spp. using several different assays that have varying sensitivities and specificities. While these assays are standard protocol (Nielsen 2002), they were developed to detect *B. abortus* in cattle, and, therefore, when they are applied to domestic or feral swine they may not accurately reflect the true prevalence of swine brucellosis. The serological tests utilized in this study do not distinguish between *B. suis* and *B. abortus* infections (Olsen 2010). Consequently, we are

Table 2. Apparent prevalence of swine brucellosis in all states where samples were collected from March 1, 2009, through December 31, 2010.

State	Total positives	# of samples collected	Apparent prevalence (%)	95% Confidence interval
Alabama	11	102	10.8	4.76–16.8
Arizona	0	68	0.0	0.00–5.34
Arkansas	11	350	3.1	1.32–4.97
California	0	264	0.0	0.00–1.43
Colorado	0	8	0.0	0.00–32.44
Florida	29	464	6.3	4.05–8.45
Georgia	5	296	1.7	0.22–3.16
Hawaii	33	229	14.4	9.86–18.96
Idaho	0	2	0.0	0.00–65.75
Illinois	0	19	0.0	0.00–16.81
Iowa	0	8	0.0	0.00–32.44
Kansas	1	142	0.7	0.00–2.0
Kentucky	0	17	0.0	0.00–18.43
Louisiana	3	136	2.2	0.00–4.67
Michigan	0	14	0.0	0.00–21.53
Mississippi	9	238	3.8	2.00–7.02
Missouri	2	201	1.0	0.00–2.37
Nebraska	0	3	0.0	0.00–56.15
Nevada	0	3	0.0	0.00–56.15
New Hampshire	0	16	0.0	0.00–19.36
New Jersey	0	9	0.0	0.00–29.91
New Mexico	0	133	0.0	0.00–2.80
New York	0	21	0.0	0.00–15.46
North Carolina	6	157	3.8	0.80–6.82
North Dakota	0	5	0.0	0.00–43.44
Ohio	0	28	0.0	0.00–12.06
Oklahoma	18	181	9.9	5.59–14.30
Oregon	0	79	0.0	0.00–4.63
Pennsylvania	0	38	0.0	0.00–9.18
South Carolina	20	173	11.6	6.80–16.33
Tennessee	0	107	0.0	0.00–3.46
Texas	11	884	1.2	0.00–2.2
Virginia	0	45	0.0	0.00–7.86
West Virginia	0	29	0.0	0.00–11.69
Wisconsin	0	10	0.0	0.00–27.75

unable to determine whether exposure comes primarily from *B. suis*, *B. abortus*, or other *Brucella* spp. Likewise, it has been shown that *Yersinia enterocolitica* infection in swine also can cause a false positive brucellosis test result (Jungersen et al. 2006). However, feral swine are typically exposed to *Y. enterocolitica* early in life, and the period of antibody cross reactivity is so short-lived (Jungersen et al. 2006, Fredriksson-Ahomaa et al. 2009) that adults would likely not have a cross-reacting antibody signature. Collecting lymph nodes and other tissues to identify the specific species of *Brucella* causing disease locally could be very useful for improving diagnostics.

Our results demonstrate that up to 14% of feral swine have been exposed to brucellosis, depending on the state. Although we estimated apparent prevalence of brucellosis by state, considerable variation existed at different geographic and temporal scales within each state. In addition, we were unable to detect any seropositive animals in 22 states. These negative results could indicate that *Brucella* spp. may truly be absent in some feral swine populations or that prevalence in these states was too low

to be detected with our sample sizes. It also is possible that sampling in some states was too spatially limited to detect a disease that appears to have a clustered distribution across the landscape. Feral swine are closely linked to resource-rich environments with permanent water sources (marshes, rivers, etc.), and family groups (sounders) are not evenly distributed across the landscape (Sparklin et al. 2009). Additionally, *Brucella* spp. have previously been detected in California and Tennessee feral swine populations (WS-National Wildlife Disease Program, unpublished data), yet, we did not identify any positive samples during this study.

Seropositive swine primarily were detected in the southern and southeastern United States and Hawaii. This regional association may simply reflect either pockets of disease or an association with the large and long-established feral swine populations (Mayer and Brisbin 2008). Data suggest that feral swine populations are expanding (Waithman et al. 1999, Gipson et al. 2006, Olsen 2010), and, while this study did not address disease spread, it has previously been demonstrated that host expansion can be accompanied by disease expansion (Daszak et al. 2000). While it is possible that the disease is expanding as feral swine populations disseminate across the landscape, linear spread may not be the rule, because feral swine are often translocated by hunters to establish new populations for sport. A similar pattern was observed when a rabies epizootic began in the mid-Atlantic region of the United States after raccoons were translocated from a rabies enzootic region in the southeastern part of the country (Rupprecht et al. 1995).

The association between *Brucella* spp. positivity in feral swine and age class revealed that older animals are more likely than juveniles to have been exposed to the pathogen. Adult animals have more opportunities over time to encounter another positive animal. Because transmission can be associated with mating (Thorne 2001), the probability of exposure likely increases once an animal is of breeding age, although, transmission is believed to occur through social contact with fluids from infected animals (Deyoe 1986). While some previous research has found that males are more likely to have been exposed to *Brucella* spp. (Stoffregen et al. 2007), other research has found equal levels of exposure in males and females (Wykoff et al. 2009), which agrees with our findings.

The relationship between season of capture and *Brucella* spp. exposure in feral swine is less clear. Animals were more likely to test positive for brucellosis during the winter, and seroprevalence was lowest during summer months; however, laboratory assays detected only antibodies and did not pinpoint when animals actually became infected. Our data indicate only that animals have been exposed and have mounted an immune response. Limited information exists on *Brucella* spp. exposure and infection in feral swine (Stoffregen et al. 2007, Wyckoff et al. 2009), but the substantial difference in seroprevalence across seasons suggests that there are periods when animals have a greater rate of exposure to *Brucella* spp. If a majority of transmission occurs while mating, then an increase in seropositivity would be expected during and directly after the breeding season. Research by Baber and Coblentz (1986) suggests that there is often some degree of breeding synchrony in feral swine populations; most breeding occurs in the fall and winter (October to March), with a small, second breeding season occurring in late summer (July to August). Breeding seasons could vary across broad geographic regions, although they are often related to photoperiod, which is less variable, or with the nutritional resources available (Baber and Coblentz 1986). The October through March breeding peak coincides with the increase in swine brucellosis detected during winter months in this study. The higher seroprevalence levels seen in adults, compared to juveniles and sub-adults, also suggests that breeding-aged animals are driving the exposure levels related to season. Juveniles and sub-adults had consistently low brucellosis seroprevalence across all seasons (<2%).

Our spatial analysis revealed several distinct disease clusters where the number of infected swine was higher than expected given background disease levels. Brucellosis clusters could be related to numerous variables, including the various laboratories conducting the diagnostics, but robust data on feral swine populations often are lacking. This makes it difficult to link disease clusters with the mechanisms driving transmission. Increased

likelihood of *Brucella* spp. infections may occur when there are large, established feral swine populations, a pattern that was seen with elk population density and *B. abortus* seroprevalence (Cross et al. 2010). High population densities could lead to increased contact rates and, consequently, increased transmission that results in disease clusters. If an infected individual is introduced to a naïve feral swine population (i.e., through human translocation), a rash of new infections could also produce a cluster of seropositive individuals. And while the spatial scan statistic can identify regions and time periods with disease exposure rates that are above background, the program cannot take into account regions or time periods that were not sampled during opportunistic collections. Consistent sampling across space and time would provide a more robust clustering analysis. Even with this uncertainty, the disease clusters demonstrate that infection is often geographically localized and not randomly distributed across the landscape. Regions that are associated with swine brucellosis clusters could use the information to inform hunters and others of the associated health risks.

Management implications

The recent geographic expansion of feral swine across the United States (Seward et al. 2004) helped to motivate this broad-scale surveillance effort on apparent prevalence of *Brucella* spp. in feral swine. Our findings suggest that: (1) the disease is present and is being transmitted in multiple regions of the continental United States and Hawaii; (2) state-level apparent prevalence values offer only a rough estimate and that finer-scale estimates reveal higher seroprevalence in localized regions, along with little to no disease in other regions; (3) adult animals and animals sampled during winter months are more likely to have detectable antibodies; and (4) clusters of disease in certain areas could mean higher risk for hunters and others who have contact with feral swine. Swine brucellosis is well-established in a number of feral swine populations, and it is important to limit further geographic spread and associated increased disease risk by implementing stricter regulations and enforcing existing ones to discourage people from translocating feral swine populations. It also is important to develop educational materials that inform the public and farmers of the potential for disease exposure. Disease control via eradication of the feral swine population, now estimated at 5 million (Pimentel 2007), may be difficult, if not impossible, to accomplish. Development of an effective oral vaccine and delivery system that can be distributed remotely to feral swine, in combination with continued efforts to reduce population sizes, may be warranted to reduce the threat of *Brucella* spp. transmission in localized, high-risk situations.

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KERRI PEDERSEN joined the Peace Corps in Guatemala after completing her B.S. degree at Oregon State University. She moved to Colorado and obtained an M.S. degree in ecology from Colorado State University. She compared the prevalence of *Salmonella* spp. and *E. coli* in urban populations of pigeons versus dairy pigeons. She worked with the Southeastern Cooperative Wildlife Disease Study in Athens, Georgia, for 2 years before moving back to Fort Collins to accept her current position as a wildlife disease biologist with the U.S. Department of Agriculture. In her current position, she is responsible for assisting with coordination of various disease surveillance activities including various feral swine diseases, Newcastle disease, a swan project, and various other tasks.

SARAH N. BEVINS is a broadly trained ecologist whose research focuses on the ecology of zoonotic disease dynamics. She graduated in 2001 from College of the Atlantic in Bar Harbor, Maine, with a B.S. degree in ecology. She received her Ph.D. degree in ecology at Colorado State University in 2007 after designing original research that documented the indirect effects that invasive vector species have on vector-borne disease transmission. She was then a postdoctoral fellow in the Colorado State University College of Veterinary Medicine and College of Natural Resources, where she was part of a National Science Foundation funded project that examined disease transmission near urban areas among bobcats, pumas, and domestic cats. Additional laboratory and field work have ranged from documenting marine mammal distributions, to small mammal trapping, and parasitology. She is currently a wildlife disease biologist with the National Wildlife Disease Program in Fort Collins, Colorado.

BRANDON S. SCHMIT'S professional interests include wildlife disease ecology, veterinary epidemiology, phenotypic plasticity, and the impacts of infectious disease on the evolution of life history traits. He grew up in Arizona and received his B.S. degree in biology at Northern Arizona University in 1993. His lifelong interest in marine ecology took him to Florida State University where he earned his M.S. degree in ecology (1997); subsequently, he worked for the Florida Department of Environmental Protection. In 2001, he began employment at Wildlife Services' National Wildlife Research Center where he worked on optimization of oral rabies vaccine (ORV) baiting strategies, development of ORV baits for skunks, and improvements in biomarkers used to monitor remote bait uptake in wildlife. In 2005, he joined Wildlife Services' National Wildlife Disease Program (NWDP). His work includes assisting in coordinating nationwide surveillance for avian influenza in wild birds and plague and tularemia monitoring in relevant wildlife species. He is involved with implementing the NWDP's comprehensive feral swine disease surveillance project, which currently focuses on >10 endemic and foreign animal diseases of importance to the domestic livestock industry and human health.

MARK W. LUTMAN grew up in a small rural town in North Dakota. He received his B.S. degree in biology from North Dakota State University (NDSU) in 1997. After completing his degree, he worked as a zookeeper for the Chahinkapa zoo, but later returned to NDSU were he received his M.S. degree in zoology in 2000. In 2001, he accepted an urban specialist position with the USDA/APHIS/Wildlife Services in Phoenix, Arizona. While in Arizona, he worked as a wildlife disease biologist in 2003. In 2007, he accepted a staff biologist position under the Wildlife Services' National Wildlife Disease Program in Fort Collins, Colorado. As a staff biologist, he is involved in various avian and feral swine diseases, as well as smaller vector-borne disease projects. His hobbies include skiing, biking, hiking, chess, and ping pong.

MICHAEL P. MILLESON is a wildlife disease biologist with the USDA Wildlife Services program in Florida. He has coordinated multiple interagency disease surveillance and monitoring projects in Florida, including early detection projects for highly pathogenic avian influenza in wild birds and classical swine fever in feral swine. Prior to his role as a wildlife disease biologist, he worked as a wildlife technician with the Wildlife Services' National Wildlife Research Center, Florida Field Station. He received his M.S. degree in wildlife ecology and conservation from the University of Florida.

CLINT T. TURNAGE received a B.S. degree from the University of Arkansas at Little Rock in 1999. After graduation, he accepted a position with the University of Tennessee as a research technician for a black bear population dynamics project on White River National Wildlife Refuge in southeast Arkansas. He later became a research associate and supervised all field operations on the project, which expanded to include the repatriation of black bears to Felsenthal National Wildlife Refuge. In 2004, he began work as a biological laboratory technician for the U.S. Department of Agriculture's Veterinary Services Brucellosis Laboratory in Little Rock, Arkansas. In 2007, he accepted a position with the U.S. Department of Agriculture Wildlife Services' National Wildlife Disease Program as a wildlife disease biologist for Arkansas. He currently lives and works in Arkansas and is married with 2 daughters.

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THOMAS J. DELIBERTO graduated from Colorado State University with a B.S. degree in wildlife biology in 1984. In 1987 he received an M.S. degree in wildlife management from Texas Tech University where he studied the nutritional ecology of white-tailed deer. In 1997, he obtained his Ph.D. degree in range science from the Utah State University and went on to earn his D.V.M. degree at Washington State University. He then worked with Wildlife Services' National Wildlife Research Center at the predation ecology and behavior field station in Logan, Utah, and, in 2001, he became the wildlife disease project leader and began researching rabies and bovine tuberculosis in wildlife. Currently, he serves as the national wildlife disease coordinator for Wildlife Services. His duties include implementation of the National Wildlife Disease Surveillance and Emergency Response System. He was the chairperson for the interagency working group that developed the U.S. strategic plan for an early detection system for highly pathogenic H5N1 avian influenza in wild migratory birds, and was on the writing team that developed the national plan for assisting states, federal agencies, and tribes in managing white-nose syndrome in bats. He currently serves on the interagency wild bird highly pathogenic avian influenza (HPAI) steering committee, the white nose syndrome executive and steering committees, the federal biosurveillance work group, and the foreign animal disease threats wildlife task force. He also serves on the board of directors for the U.S. Animal Health Association and The Wildlife Society's wildlife disease working group and on the advisory council for the American Association of Wildlife Veterinarians. Additionally, he coordinates wildlife disease surveillance and capacity building projects in China, Southeast Asia, Indonesia, Greenland, Mexico, Kenya, Uganda, and the Ukraine.